

Hyperventilation

CO2 - medical research and medical references

CO2 (carbon dioxide) health effects and benefits for the human body are innumerable in spite of environmental concerns related to global warming. Life was born and had existed for millennia at very high CO2 content in air (up to about 7-12% when the first lungs were evolving). Note that large CO2 concentrations produce adverse effects and pure CO2 is a toxic gas to breathe. We are focused on typical or physiological CO2 levels ranging from about 20 to 50 mm Hg or from about 2.7 to 7.5%.

Normal level of CO2 in the lungs and arterial blood (40 mm Hg or about 5.3% at sea level) is imperative for normal health.

Hypocapnia (CO2 deficiency) is a normal finding for chronic diseases due to prevalence of chronic hyperventilation among the sick. Furthermore, as we discovered before, over 90% of modern people ("normal subjects") are hyperventilators (see [Hyperventilation Table](#) with over 20 medical research studies). Hence, chronic hypocapnia is very common for modern man.

Hyperventilation in the sick and normal breathing rates in healthy subjects

What do we know about breathing rates in people with chronic diseases? Do healthy people have normal minute ventilation rates?

Minute ventilation rates (chronic diseases)

Condition	Minute ventilation	Number of people	All references or click below for abstracts
Normal breathing	6 L/min	-	Medical textbooks
Healthy Subjects	6-7 L/min	>400	Results of 14 studies
Heart disease	15 (±4) L/min	22	Dimopoulou et al, 2001
Heart disease	16 (±2) L/min	11	Johnson et al, 2000
Heart disease	12 (±3) L/min	132	Fanfulla et al, 1998
Heart disease	15 (±4) L/min	55	Clark et al, 1997
Heart disease	13 (±4) L/min	15	Banning et al, 1995
Heart disease	15 (±4) L/min	88	Clark et al, 1995
Heart disease	14 (±2) L/min	30	Buller et al, 1990
Heart disease	16 (±6) L/min	20	Elborn et al, 1990
Pulm hypertension	12 (±2) L/min	11	D'Alonzo et al, 1987

Cancer	12 (\pm 2) L/min	40	Travers et al, 2008
Diabetes	12-17 L/min	26	Bottini et al, 2003
Diabetes	15 (\pm 2) L/min	45	Tantucci et al, 2001
Diabetes	12 (\pm 2) L/min	8	Mancini et al, 1999
Diabetes	10-20 L/min	28	Tantucci et al, 1997
Diabetes	13 (\pm 2) L/min	20	Tantucci et al, 1996
Asthma	13 (\pm 2) L/min	16	Chalupa et al, 2004
Asthma	15 L/min	8	Johnson et al, 1995
Asthma	14 (\pm 6) L/min	39	Bowler et al, 1998
Asthma	13 (\pm 4) L/min	17	Kassabian et al, 1982
Asthma	12 L/min	101	McFadden & Lyons, 1968
Sleep apnea	15 (\pm 3) L/min	20	Radwan et al, 2001
Liver cirrhosis	11-18 L/min	24	Epstein et al, 1998
Hyperthyroidism	15 (\pm 1) L/min	42	Kahaly, 1998
Cystic fibrosis	15 L/min	15	Fauoux et al, 2006
Cystic fibrosis	10 L/min	11	Browning et al, 1990
Cystic fibrosis*	10 L/min	10	Ward et al, 1999
CF and diabetes*	10 L/min	7	Ward et al, 1999
Cystic fibrosis	16 L/min	7	Dodd et al, 2006

Cystic fibrosis	18 L/min	9	McKone et al, 2005
Cystic fibrosis*	13 (\pm 2) L/min	10	Bell et al, 1996
Cystic fibrosis	11-14 L/min	6	Tepper et al, 1983
Epilepsy	13 L/min	12	Esquivel et al, 1991
CHV	13 (\pm 2) L/min	134	Han et al, 1997
Panic disorder	12 (\pm 5) L/min	12	Pain et al, 1991
Bipolar disorder	11 (\pm 2) L/min	16	MacKinnon et al, 2007
Dystrophia myotonica	16 (\pm 4) L/min	12	Clague et al, 1994

This data explain the pathological changes and high prevalence of chronic disorders (or diseases of civilization), due to hyperventilation, in modern population. Since modern people breathe about 2 times more than the medical norm, they usually suffer from low CO₂ values in the arterial blood (ventilation-perfusion mismatch is not a very common condition). Carbon dioxide is a potent dilator of blood vessels (vasodilator) and is crucial for the Bohr effect (O₂ transport from red blood cells to tissues). As a result, modern people experience reduced oxygen levels in the brain, heart, kidneys and all other vital organs. Cell hypoxia causes or favors inflammatory conditions, production of free radicals and suppression of the immune system. Apart from these effects, there are devastating effects of hyperventilation syndrome on the brain due to hypocapnia due to lack of CO₂ that has calming or sedative properties. These and other hyperventilation-related physiological effects promote pathological changes and advance of chronic health problems.

Is hyperventilation common on ordinary people?

Chronic hyperventilation is very common for ordinary people (or "normal subjects") these days. Their average minute ventilation values are much greater than the normal value, which used to be the norm about 80-100 years ago.

How can we increase cell oxygenation or cell oxygen levels? What is the ideal breathing pattern that provides vital organs (the brain, heart, kidneys, liver and so on) with maximum oxygen levels? How should we breathe day and night for higher brain oxygenation?

When we breathe more than the norm (and this is a case for over 90% of modern cell oxygen level is reduced and we suffer from cell hypoxia. Indeed, during normal breathing, our arterial blood has nearly maximum oxygen saturation. Hence, the prime effect of overbreathing is reduced CO₂ content in the lungs, blood and other body cells and tissues. Medical studies have found that hyperventilation reduces cell oxygen level in the following vital organs:

- brain (Brown, 1953; Kennealy et al, 1980; Liem et al, 1995; Lum, 1975; Lum, 1982; Macey et al, 2007; Litchfield, 2003; Santiago & Edelman, 1986; Skippen et al, 1997; Starling & Evans, 1968; Tsuda et al, 1987)
- heart (Foëx et al, 1979; Karlsson et al, 1994; Okazaki et al, 1991; Okazaki et al, 1992; Wexels et al, 1985)
- liver (Fujita et al, 1989; Hughes et al, 1979; Okazaki, 1989)
- kidneys (Karlsson et al, 1994; Okazaki, 1989)
- spleen (Karlsson et al, 1994)
- colon (Guzman et al, 1999)

- systemic or body tissues in general (Laffey & Kavanagh, 2002; Nunn, 1987).

For most people, low cell oxygen levels are produced due to 2 effects: constriction of arteries and arterioles (since CO₂ is a most potent vasodilator) and the suppressed Bohr effect (less oxygen is released in tissues due to increased affinity of oxygen to red blood cells caused by hypocapnia). These are the main effects leading to reduced cells oxygen content mentioned by many physiologists and doctors (see the quotes and references below). In particular, hyperventilation reduces brain oxygenation.

Vasodilation- kapillärvidgande effekt

Vasodilation(expansion of arteries and arterioles). As physiological studies found, hypocapnia (low CO₂ concentration in the arterial blood) constricts blood vessels and leads to decreased perfusion of all vital organs

The Bohr effect was first described in 1904 by the Danish physiologist Christian Bohr (father of physicist Niels Bohr). This law can be found in modern medical textbooks on physiology. The Bohr effect states that arterial hypocapnia will cause reduced oxygen release in tissue capillaries.

Cells Oxygen Levels are controlled by alveolar CO₂ and breathing. Hyperventilation, regardless of the arterial CO₂ changes, causes alveolar hypocapnia (CO₂ deficiency) that leads to cell hypoxia (low cells oxygen concentrations)

Oxygen Transport, therefore, depends on breathing and these 2 effects (Vasoconstriction-Vasodilation and the Bohr effect) are part of 2 diagrams that summarizes influences of hypocapnia (low CO₂ content in the blood and cells) on circulation and O₂ delivery.

- **Free Radicals Generation** take place due to anaerobic cell respiration caused by cell hypoxia. Hence, antioxidant defenses of the human body are also regulated by CO₂ and breathing, as these medical studies have found.

Inflammatory Response are also, in a long run (chronic inflammation), controlled by breathing since hypoxia leads to or intensifies inflammation. Therefore, hyperventilation naturally promotes inflammatory health problems and CO₂ is the key anti-inflammatory agent.

- **Nerve Stabilization** is due to calmativ or sedative effects of carbon dioxide on nervous cells. Lack of CO₂ in the brain leads to "*spontaneous and asynchronous firing of neurons*" (medical quote) "inviting" virtually all mental and psychological abnormalities ranging from panic attacks and seizures to sleeping problems, depression and schizophrenia.

Muscle relaxation or relaxation of muscle cells is normal at high CO₂, while hypocapnia causes muscular tension, poor posture and, sometimes, aggression and violence.

Brochodilation - dilation of airways: bronchi and bronchioles by carbon dioxide, and their constriction due to hypocapnia.

References and quotes (Cells Oxygen and Hyperventilation/Hypocapnia)

Section "Physiologic and Biochemical Consequences"

"There is a decrease in cerebral oxygen tension on the basis of both the Bohr effect and the decreased cerebral blood flow."
Brashear RE, **Hyperventilation Syndrome**, Lung, 1983, 161: p. 257-273.

Division of Pulmonary Medicine, Indiana University School of Medicine, University Hospital, Indianapolis, Indiana 46223, USA

Section "Neurologic Effects of Hypocapnia"

"Systemic hypocapnia results in cerebrospinal fluid alkalosis, which decreases cerebral blood flow, cerebral oxygen delivery, and to a lesser extent, cerebral blood volume."

Laffey JG & Kavanagh BP, Hypocapnia, New England Journal of Medicine 2002, 347(1) 43-53.

"Because both hypocapnia and alkalosis cause a leftward shift of the oxyhemoglobin dissociation curve, off-loading of oxygen at the tissue level is restricted. In addition, hypocapnia causes systemic arterial vasoconstriction, decreasing the global and

regional oxygen supply and compounding the reduction in the delivery of oxygen to tissue."
Laffey JG & Kavanagh BP, Hypocapnia, New England Journal of Medicine 2002, 347(1) 43-53.

"First consider the immediate effects of hypocapnia. The most striking direct effect is on the cerebral circulation. Carbon dioxide is the most important regulator of cerebral vascular tone. Hypocapnia causes immediate vasoconstriction leading to cerebral hypoxia."

Lum LC, Hyperventilation: The Tip and the Iceberg, Journal of Psychosomatic Research, 1975, Vol. 19, pp. 375-383.

"Furthermore, carbon dioxide controls the calibre of cerebral arteries. Hypocapnia causes vasoconstriction, and hence cerebral hypoxia. This hypoxia is augmented by a shift to the left of the haemoglobin dissociation curve for oxygen (Bohr effect), which diminishes both the amount and the rate of transfer of oxygen to tissues."

Lum LC, Hyperventilation and Anxiety State, Journal of the Royal Society of Medicine, 1981 (74) 1-4

Blood pH regulation and regulation of other bodily fluids

Changes in carbon dioxide and breathing cause immediate and long-term effects of blood pH. They are not necessarily the same. The immediate effects are simple: higher CO₂ content causes blood acidification and pH decrease, while reduced carbon dioxide levels increase blood pH often causing death in the critically ill (see a review of medical studies below). Long term effects depend on the direction of change (moving closer to normal breathing or not), genetic factors, existing pathologies, diet, physical exercise, thermoregulation, and many other parameters.

CO₂ gas, when dissolved in blood, is the second largest group of negative ions of blood plasma. Hence, breathing directly affects blood pH. In its turn, blood pH is tightly monitored within a very narrow range (from about 7.3 to 7.5) by the group of nerve cells located in the medulla oblongata in order to have normal body biochemistry. The same nerve cells control breathing by through several independent mechanisms, including peripheral and central CO₂ and O₂ chemoreceptors.

It is not a surprise that even mildly sick patients suffer from blood pH abnormalities due to breathing since they breathe about 2-3 times more than the medical norm. For review of 34 medical studies

Hence, arterial CO₂, carbon dioxide, through several independent biochemical mechanisms can influence blood pH and causes respiratory alkalosis in patients with chronic diseases.

CO₂, hypocapnia and viscosity of blood

CO₂ also influences viscosity of blood. Acute hyperventilation and arterial hypocapnia makes blood more viscous. This effect is a part of the fight-and-flight response (an immediate reaction to stress). While useful in a short run to prevent blood losses due to bleeding, increased blood viscosity produces a large strain on the heart muscle and causes other negative effects leading to, for example, thrombosis (formation of a blood clot).

Dr. K. P. Buteyko and his colleagues also found that CO₂ controls and regulates composition and properties of many all other bodily fluids, including secretions of the stomach, composition and properties of saliva and mucus, pH of the urine. For example, for most people, in conditions of hyperventilation, stomach and urinary pH become too low (too acidic) promoting development of gastritis and ulcers, or urinary stones.

On the influence of CO₂ on the viscosity of the blood

Russell Burton-Opitz

From the Physiological Laboratory of Columbia University, at the College of Physicians and Surgeons.

Abstract

It has been proved by the author¹ that the blood in the veins possesses a somewhat greater viscosity than the blood in the arteries. As this difference is caused no doubt by the greater amount of CO₂ present in the venous blood, it became of some consequence to determine whether the arterial blood could be made to assume a greater viscosity by increasing its CO₂ content.

The dogs used in these experiments received alternately a supply of normal air and air charged with CO₂. During the period of inhalation of the air plus CO₂ the arterial blood showed a somewhat greater viscosity than during the time when the animal breathed normal air. The changes appeared very promptly, but were never very conspicuous. The specific gravity of the blood pursued a course parallel to that of the viscosity.

Experimental Medicine journal: 1903, i, p. 23.

About 200 Russian and Soviet Buteyko doctors have accumulated valuable clinical experience in this area since they have treated more than 200,000 people. These medical professionals discovered that there are several practical details that are crucial for successful treatment of chronic hyperventilation:

- 1) Understanding that breathing less at rest delivers more oxygen to body cells.
- 2) Constant commitment to breathing normalization since the purpose of training is to change one's automatic (or basal) breathing pattern.
- 3) Daily measurement of the progress: either using devices (e.g., capnometers, plastic bag to measure minute ventilation - supplied with the Samozdrav breathing device) or the body oxygen test.
- 4) Understanding that even short episodes of hyperventilation (e.g., for 1-2 hours due to sleep heavy breathing effect, overeating, stress) produce serious enough damage to reverse all progress achieved during the remaining part of the day (see links related to oxidative stress caused by hypocapnia - low CO₂).

Here are some lifestyle factors that are important (or essential) to address for successful hyperventilation treatment. Depending on personal lifestyle and environmental factors, there are dozens more lifestyle factors that can have either a positive or negative effect on breathing retraining.

Among arterial dilators, natural vasodilation agent CO₂ is probably the most powerful chemical. The vasodilation effect is present in healthy people due to normal arterial CO₂ concentration. According to Dr. M. Kashiba, MD and his medical colleagues from the Department of Biochemistry and Integrative Medical Biology, School of Medicine, Keio University in Tokyo, CO₂ is a "*potent vasodilator*" (Kashiba et al, 2002), while Dr. H. G. Djurberg and his team from the Department of Anaesthesia, Armed Forces Hospital, in Riyadh, Saudi Arabia suggested that "*Carbon dioxide, a most potent cerebral vasodilator...*" (Djurberg et al, 1998).

Effects of arterial hypocapnia caused by hyperventilation

Based on tens medical research studies, we established presence of the [hyperventilation syndrome](#) in the sick due to elevated minute ventilation. What are the effects of chronic hyperventilation of the human body? One of the central [carbon dioxide effects](#) is constriction of arteries and arterioles due to CO₂ deficiency. Instead of vasodilation, arteries and arterioles get constricted.

Medical publications related to CO₂-induced vasodilation

Dr. K. P. Buteyko and his colleagues found vasoconstrictive effects of hypocapnia (CO₂ deficiency) on arteries and peripheral blood vessels (Buteyko et al, 1964a; Buteyko et al, 1964b; Buteyko et al, 1964c; Buteyko et al, 1965; Buteyko et al, 1967), while additional CO₂ causes vasodilation, which is a normal state of arteries and arterioles.

As western physiological studies found, vasodilation requires normal arterial CO₂ concentration, while hypocapnia (low CO₂ concentration in the arterial blood) decreased perfusion of the following organs:

- brain (Fortune et al, 1995; Karlsson et al, 1994; Liem et al, 1995; Macey et al, 2007; Santiago & Edelman, 1986; Starling & Evans, 1968; Tsuda et al, 1987),
- heart (Coetzee et al, 1984; Foëx et al, 1979; Karlsson et al, 1994; Okazaki et al, 1991; Okazaki et al, 1992; Wexels et al, 1985),
- liver (Dutton et al, 1976; Fujita et al, 1989; Hughes et al, 1979; Okazaki, 1989),
- kidneys (Karlsson et al, 1994; Okazaki, 1989),
- spleen (Karlsson et al, 1994),
- colon (Gilmour et al, 1980).

Some abstracts from these studies

References for CO₂ vasodilation effect

Buteyko KP, Odintsova MP, Dyomin DV, *Hyper- and Hypoxemia Effects on the Peripheral Vascular Tone*, Materials of the Second Siberian Research Conference of Therapists, Irkutsk, 1964a.

Buteyko KP, Dyomin DV, Odintsova MP, *Regressive Analysis in Differentiating Aerated Blood Gas Component Effects on Peripheral Arteriole Functional Conditions*, Materials of the Second Siberian Research Conference of Therapists, Irkutsk, 1964b.

Buteyko KP, Zhuk EA, Mikaelyan AL, *Electrocardiogram for Isolated Aortal Stenosis*, *Cardiologiya (Cardiology, USSR)*, 1964c, N 2, p. 67.

Buteyko KP, Dyomin DV, Odintsova MP, *Ventilation of the Lungs and Arterial Vascular Tone Interconnection in Patients with High Blood Pressure and Angina Pectoris*, *Phyziologichny Zhurnal (Physiological Magazine, Ukrainian SSR)* 1965. V. 11, N 5 (in Ukrainian).

Buteyko KP, Odintsova MP, Dyomin DV, *Hyper- and Hypoxemia Effects on the Arterial Vascular Tone*. *Sovetskaya Meditsina (Soviet Medicine)*, 1967, N3, p.44-49.

Coetzee A, Holland D, Foëx P, Ryder A, Jones L, *The effect of hypocapnia on coronary blood flow and myocardial function in the dog*, *Anesthesia and Analgesia* 1984 Nov; 63(11): p. 991-997.

Dutton R, Levitzky M, Berkman R, *Carbon dioxide and liver blood flow*, *Bull Eur Physiopathol Respir.* 1976 Mar-Apr; 12(2): p. 265-273.

Gilmour DG, Douglas IH, Aitkenhead AR, Hothersall AP, Horton PW, Ledingham IM, *Colon blood flow in the dog: effects of changes in arterial carbon dioxide tension*, *Cardiovasc Res* 1980 Jan; 14(1): 11-20.

Foëx P, Ryder WA, *Effect of CO₂ on the systemic and coronary circulations and on coronary sinus blood gas tensions*, *Bull Eur Physiopathol Respir* 1979 Jul-Aug; 15(4): p.625-638.

Fortune JB, Feustel PJ, deLuna C, Graca L, Hasselbarth J, Kupinski AM, *Cerebral blood flow and blood volume in response to O₂ and CO₂ changes in normal humans*, *J Trauma.* 1995 Sep; 39(3): p. 463-471.

Fujita Y, Sakai T, Ohsumi A, Takaori M, *Effects of hypocapnia and hypercapnia on splanchnic circulation and hepatic function in the beagle*, *Anesthesia and Analgesia* 1989 Aug; 69(2): p. 152-157.

Hashimoto K, Okazaki K, Okutsu Y, *The effects of hypocapnia and hypercapnia on tissue surface PO₂ in hemorrhaged dogs* [Article in Japanese], *Masui* 1989 Oct; 38(10): p. 1271-1274.

Henderson Y, *Acapnia and shock - I. Carbon dioxide as a factor in the regulation of the heart rate*, *American Journal of Physiology* 1908, 21: p. 126-156.

Hughes RL, Mathie RT, Fitch W, Campbell D, *Liver blood flow and oxygen consumption during hypocapnia and IPPV in the greyhound*, *J Appl Physiol.* 1979 Aug; 47(2): p. 290-295.

Karlsson T, Stjemström EL, Stjemström H, Norlén K, Wiklund L, *Central and regional blood flow during hyperventilation. An experimental study in the pig*, *Acta Anaesthesiol Scand.* 1994 Feb; 38(2): p.180-186.

Liem KD, Kollée LA, Hopman JC, De Haan AF, Oeseburg B, *The influence of arterial carbon dioxide on cerebral oxygenation and haemodynamics during ECMO in normoaxemic and hypoxaemic piglets*, *Acta Anaesthesiol Scand Suppl.* 1995; 107: p.157-164.

Litchfield PM, *A brief overview of the chemistry of respiration and the breathing heart wave*, *California Biofeedback*, 2003 Spring, 19(1).

Macey PM, Woo MA, Harper RM, *Hyperoxic brain effects are normalized by addition of CO₂*, *PLoS Med.* 2007 May; 4(5): p. e173.

McArdle WD, Katch FI, Katch VL, *Essentials of exercise physiology* (2-nd edition); Lippincott, Williams and Wilkins, London 2000.

Okazaki K, Okutsu Y, Fukunaga A, *Effect of carbon dioxide (hypocapnia and hypercapnia) on tissue blood flow and oxygenation of liver, kidneys and skeletal muscle in the dog*, Masui 1989 Apr, 38 (4): p. 457-464.

Okazaki K, Hashimoto K, Okutsu Y, Okumura F, *Effect of arterial carbon dioxide tension on regional myocardial tissue oxygen tension in the dog* [Article in Japanese], Masui 1991 Nov; 40(11): p. 1620-1624.

Okazaki K, Hashimoto K, Okutsu Y, Okumura F, *Effect of carbon dioxide (hypocapnia and hypercapnia) on regional myocardial tissue oxygen tension in dogs with coronary stenosis* [Article in Japanese], Masui 1992 Feb; 41(2): p. 221-224.

Santiago TV & Edelman NH, *Brain blood flow and control of breathing*, in Handbook of Physiology, Section 3: The respiratory system, vol. II, ed. by AP Fishman. American Physiological Society, Bethesda, Maryland, 1986, p. 163-179.

Starling E & Lovatt EC, *Principles of human physiology*, 14-th ed., 1968, Lea & Febiger, Philadelphia.

Tsuda Y, Kimura K, Yoneda S, Hartmann A, Etani H, Hashikawa K, Kamada T, *Effect of hypocapnia on cerebral oxygen metabolism and blood flow in ischemic cerebrovascular disorders*, Eur Neurol. 1987; 27(3): p.155-163.

Wexels JC, Myhre ES, Mjøs OD, *Effects of carbon dioxide and pH on myocardial blood-flow and metabolism in the dog*, Clin Physiol. 1985 Dec; 5(6): p.575-588.